CLAIMS

We claim:

1) A method of inhibiting the proliferation of a eukaryotic cell whose growth is stimulated by β-catenin-mediated gene transcription, comprising contacting said cell with:

a) a non-endogenous source of RXR nuclear receptor protein, and

b) a therapeutically effective amount of an agonist of said RXR protein.

2) The method of claim 1 wherein said RXR agonist is not an agonist of an RAR nuclear receptor.

3) The method of claim 2 wherein said RXR protein is an RXRα protein.

4) The method of claim 1 wherein said RXR protein is expressed within said cell by an expression vector.

5) The method of claim 4 wherein said expression vector is a viral expression vector.

6) The method of claim 5 wherein said expression vector is selected from the group consisting of an adenovirus-derived expression vector, an adeno associated virus-derived expression vector and a retrovirus –derived expression vector.

7) The method of claim 6 wherein said expression vector is an adenovirus-derived expression vector.

8) The method of claim 4 in which said expression vector is injected into said cell.

9) The method of claim 1 in which said cell is a colon cell and said non-endogenous source of RXR protein is provided to said cell by means of oral or rectal administration.

10) The method of claim 9 in which said RXR ligand is contacted with said cell by systemic administration.

11) The method of claim 1 wherein said cell is a cancer cell.

12) The method of claim 11 wherein said cancer cell is a colon cancer cell.

13) A method for determining whether a test compound is an RXR agonist comprising administering said test compound to a cell which expresses RXR and β-catenin, and determining whether β-catenin is degraded in response to the addition of said test compound, wherein the degradation of said β-catenin indicates that said test compound is an RXR agonist.